# INTRAMURAL VESSELS, ACID MUCOPOLYSACCHARIDES AND LIPIDES IN THE WALL OF AORTA ABDOMINALIS AND ITS VISCERAL BRANCHES

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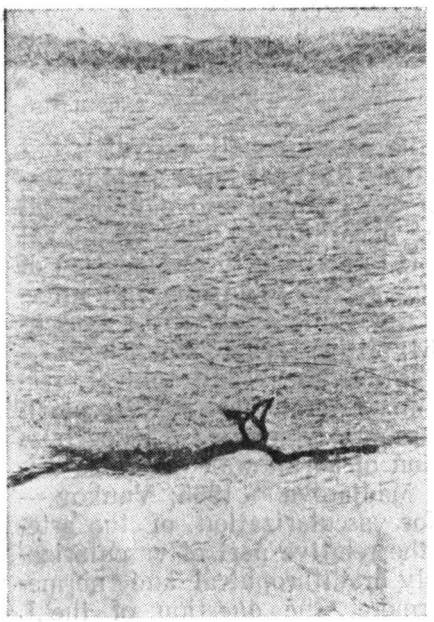
The investigations of vascularization of the arterial wall and distribution of acid mucopolysaccharides (AM) and lipides (L) in it have certain significance for revealing out the mechanisms of age changes and pathological deviations of arteries with various structure. According to bibliographical data the distribution of AM in the vessel wall is in relation to the location of the intramural vessels there. The larger amount of AM in intima and internal parts of media of aorta and large vessels is a possible result of the avascularization of these parts (Bertelsen — 1963; 1964; Mitin — 1966; Holmskaya — 1973). However, the authors explain and determine the braditrophical, respectively avascularizated part of the vessel, due to their data for vascularization of aorta, i. g. the internal $^2/_3$ of media are avascularizated (Linzbach — 1959). Principally, the degree of vascularization of aortal wall is quite different in its various regions (Vankov, Gjurov, Madjarova — 1965; Vankov — 1971; 1973), therefore, the insufficient data for vascularization of the arterial vessels of man do not define thoroughly the relative part of vascularizated and non-vascularizated zones, resprectively bradithrophical and normathrophical zones of the arterial walls. Furthermore, the question of the L amount in the structural elements and vascularization of the wall is still far from a detailed analysis.

## Materials and methods

The structure and vascularization of Aorta abdominalis and its main branches: truncus celiacus (and its branches), A. mesenterica sup., A. mesenterica inf. and A. renalis were studied with 10 human objects, aged 3—88 years. The distribution of AM and L in the cited vessels was studied with 4 of these objects, aged 27, 35, 52, 72 years, all males, died out of accidents. 5—10 hours after death the material was taken for the purposes of our investigation. Staining of the preparations with haematoxilin-eosin, orcein, alcyan-blau, alcyan-blau with background, toluidin-blue with pH 2.8, 3.5, 5.0, Sudan-schwarz and oil-red was performed. Morphometrical method determining the mean thickness of intima and total size was also applied. AM were measured according to the intensity of the reaction by 1 to 4 plus (+). The location and way of sedimentation of L in the wall was studied too.

**Results and discussion**

Our data show that in distal direction the vascularizated part of the media of A. abdominalis decreases its size and is located only in the outer $^1/_3$ (fig. 1). The terminal vessel formations in the media have a set-like type with various structural complexity due to the depth of their enter (fig. 2).



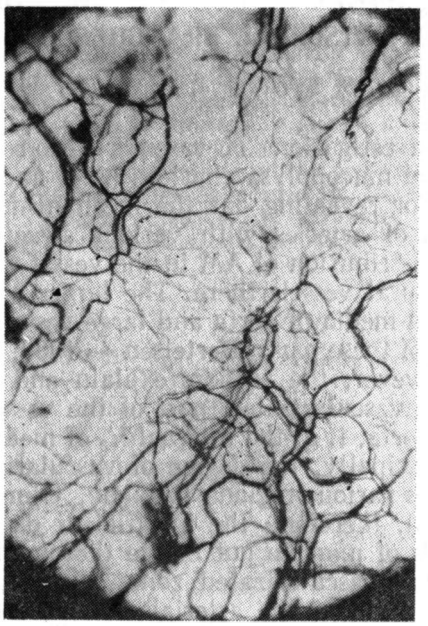


Fig. 1

Fig. 2

The arteries of muscular type in the abdominal cavity are usually vascularizated in the ranges of the adventitia and the degree of approaching the media is in a direct relation of its thickness and that of the intima (vessel wall itself). Partical enter the media of some intramural vessels is established only with a thicker arterial wall. Therefore, a conclusion can be made, that the arteries of muscular type require such depth of intramural vessels in the wall as the thickness of it allows; these data are in coordination with the previous ones of Vankov and Marinov (1973, 1977). The various forms and number of the terminal vessel formations in the media of arteries of elastic or muscular type shows that the degree of vascularization of the wall is determined by its tissue components.

The staining with alcyan-blue and toluidin-blue at pH — 5.0 detects all basic fractions of AM in the vessel wall. Our data prove that the amount and distribution of AM in the arterial wall is directly related to its structure and vascularization. Large accumulated masses of AM in arterial vessels can be established in their intact thickened intima, near to the internal elastic membrane and also in the internal parts of the media itself .The amount of AM in the thickened intima is a possible reason for their participation in the fibrillogenesis which takes place exactly here. As for the increased quantity of

AM in the neighbouring regions of internal elastic membrane with thicker intima, it is very probably to be due to an expressed anaerobial glycogenolysis based on the poorer supply with oxygen through the vessel lumen. The accumulation of AM in the most inner parts of the media can be attributed

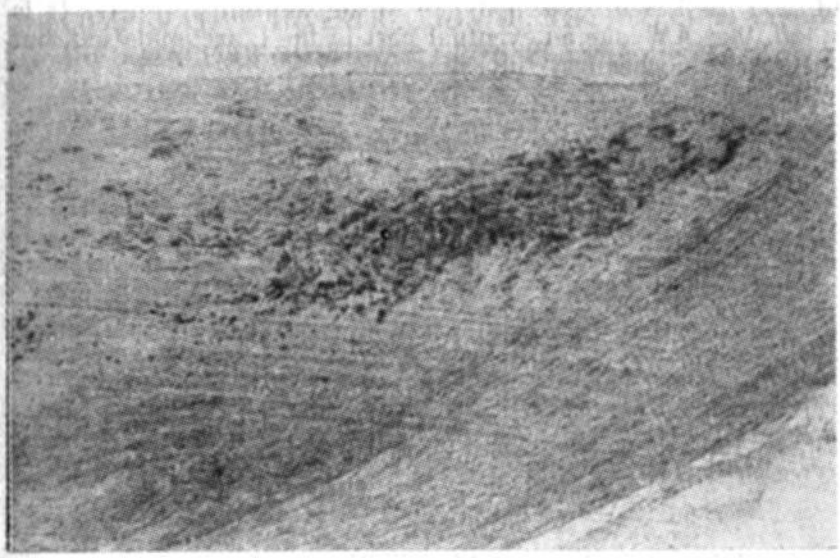


Fig. 3

also to the increased anaerobial glycogenolysis due to the far location from the intramural vessels. The less amount of AM, even absence, in the destructed intima or in regions of L infiltrations can be explained by less dynamic metabolical processes, respectively anaerobial glycogenolysis.

Our data show certainly a considerable relation between the distribution of AM and location of the intramural vessels. AM amount is bigger in all avascularizated zones of the wall: beginning from the internal border of vascularizated zones in the direction of the inner elastic membrane it is reliably increased. The contraversal direction, from intima towards adventitia, AM quantity decreases to slight, suspective or even absent values nearer the inner border of the vascularizated layer: for the aorta — its outer $^1/_3$; for certain thinwall arteries of muscular type positive reaction is established until the fibro-elastic layer (including it) and the fact can be explained by this that intramural vessels of those arteries usually do not reach the adventitial fibro-elastic layer.

The process of L accumulation in arterial wall, according to our data, begins in the considerably thicker intima, specially in its profound layers, just near to the inner elastic membrane (fig. 3). This fact proves that L come to the cited membrane from the lumen. L presence in media can be detected only if this membrane is destructed. All that confirms the filtration theory for the origin of L accumulations in the arterial wall. Something more — the accumulation of L and forming of L agglomerates and plaques in the profound layers of thickened intima and internal parts of media is highly correlated

with the AM location; these compounds are accumulated in highest degree exactly in the cited places. The correlation can be explained by the fact that the aforementioned zones in thickened intima are farest from the oxygen supplies (intramural capillaries) and the blood from the arterial lumen. The oxygen insufficiency is a possible reason for AM accumulation due to anaerobial glycolysis, but also for L sedimentation due to metabolism disorders. It is very probably too, that AM take an eventual part in the L accumulation, but this requires further biochemical investigations.

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## ВНУТРИСТЕНОЧНЫЕ СОСУДЫ, КИСЛЫЕ МУКОПОЛИСАХАРИДЫ И ЛИПИДЫ СТЕНКИ БРЮШНОЙ АОРТЫ И ЕЕ ВНУТРЕННИХ ВЕТВЕЙ

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## РЕЗЮМЕ

Изучены брюшная аорта и ее внутреностные ветви — чревный ствол, селезеночная, печеночные, брыжеечные и почечные артерии. В стенке брюшной аорты внутристеночные сосуды формируют сосудистое сплетение в адвентиции, от которого одиночные капиллярные петли иногда проникают во внешнюю часты медии. В стенке внутренностных ветвей брюшной аорты внутристеночные сосуды располагаются преимущественно в адвентиции. С возрастом они проникают в фиброэлластический слой и плотно прилегают к наружному слою медии. Иногда одиночные капилляры проникают в медию. Реакция на кислые мукополисахариды в утолщенной интиме и возле места накопления липидов становится более интенсивной; ее интенсивность уменьшается или становится отрицательной в области липидных бляшек. Накопление липидов в стенке брюшной аорты наблюдается еще у молодых индивидов. В зрелом и пожилом возрасте липидные накопления наблюдаются и в утолщенных участках интими всех исследованных нами артерии.